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Case report

Spontaneous resolution of acute cranial subdural hematomas

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Abstract

Acute cranial subdural hematoma (SDH) represents a common consequence of traumatic brain injury. The vast majority of acute SDHs larger than 10 mm in thickness require immediate surgical evacuation. In rare occasions, however, spontaneous resolution may occur. In our current communication, we present four cases of spontaneous resolution of acute cranial SDH. Further more, the proposed theories explaining spontaneous resolution of acute SDH, as well as, clinical parameters and imaging characteristics that might predict such phenomenon, are also reviewed. The possibility of spontaneous resolution of an acute SDH, although remote, may impact the decision making process regarding the management of these patients under certain conditions.
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1. Introduction

Acute subdural hematomas (SDH) represent a common consequence of head injuries. Emergent surgical evacuation is recommended in the vast majority of the acute SDH cases and definitely in those greater than 10 mm in thickness [1–4]. Contrariwise, surgical decompression of a thin rim acute SDH of 3 mm or less in diameter is unlikely to improve the patient's condition [4–6]. However, a controversial group of patients regarding their surgical or conservative management are those with an acute SDH of 5–10 mm in thickness with Glasgow Coma Scale score of 9–13 [4]. Moreover, in rare occasions spontaneous resolution of an acute SDH has been reported [7–29]. The time required for spontaneous resolution of the acute SDH in the previously reported cases ranged between a few hours and a few days after injuries [7–19,22–28]. A similar phenomenon of spontaneous resolution has been well described in patients with chronic SDHs [30–32].

In our current communication, we report a series of four adult patients with post-traumatic acute SDH, which were spontaneously resolved. With this opportunity, we review the pertinent literature with emphasis on the pathophysiologic mechanisms implicated in the resolution of acute SDH.

1.1. Case 1

Following a high speed motor vehicle accident, a 29-year-old male was transferred to our institution with GCS score of 8. The patient's laboratory results were within normal limits. A head CT scan, obtained upon admission, showed left fronto-parietal subarachnoid hemorrhage with no evidence of SDH.

An intra-parenchymal intracranial pressure (ICP) monitor was inserted in the left frontal area. The patient was admitted to the neuro-intensive care unit.

Approximately 6 h later another head CT scan was obtained, which demonstrated a 1.8 cm left-sided fronto-temporal SDH with a 2 cm midline shift (Fig. 1). The patient's neurological status, however, was unchanged. His ICP was approximately 40 mmHg while his cerebral perfusion pressure was 60–70 mmHg. In consideration of emergent surgical

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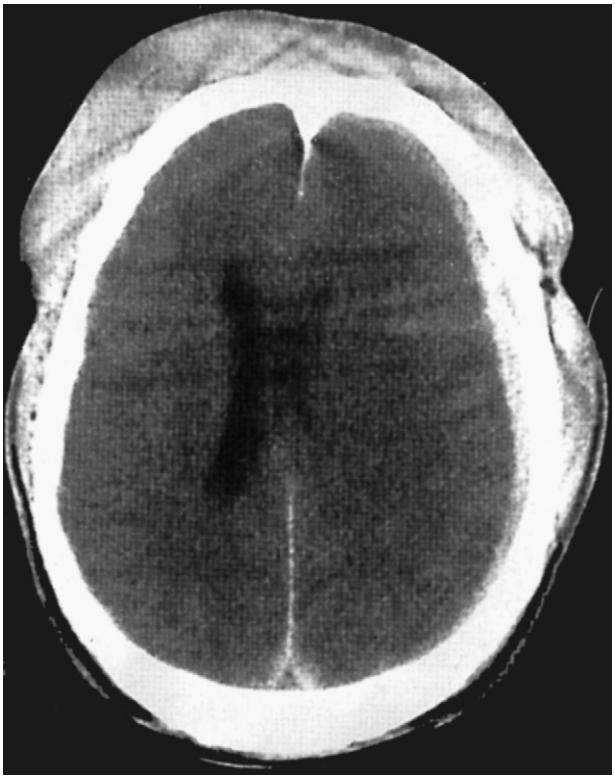

Fig. 1. Head CT scan obtained 6 h after admission, demonstrates a left-sided acute subdural hematoma (1.8 cm in its largest diameter). Note also the effacement of the left lateral ventricle and the midline structure shift.

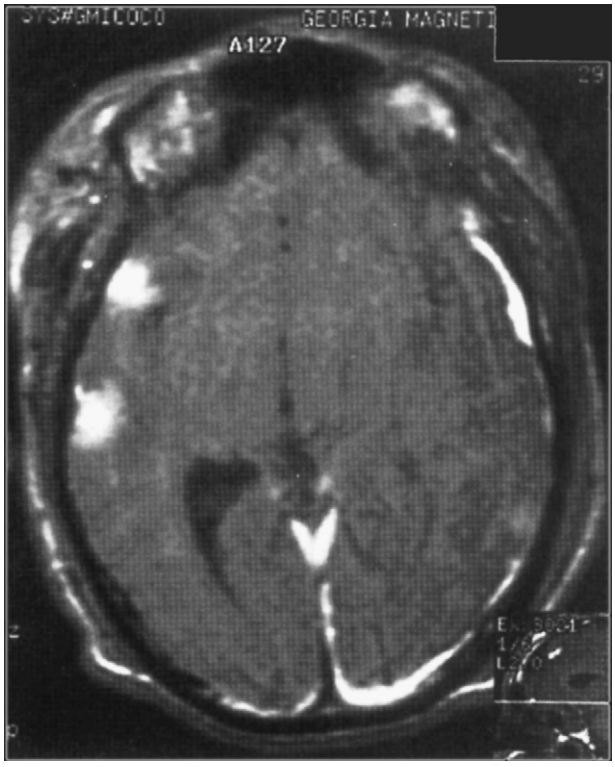

Fig. 3. Brain MRI (axial T_1 WI) demonstrates redistribution of blood in the subdural space formatting a thin layer.

evacuation of the SDH his clotting studies were re-evaluated at that time and were found to be markedly abnormal (INR >2.8). It was elected to monitor the patient closely in an attempt to correct his abnormal clotting studies rather than embarking on immediate surgical intervention. Over the following 12 h, the patient's hematological situation deteriorated and he was noted to display fulminant disseminated intravascular coagulopathy despite all efforts to correct it. To assess the intracranial situation, another head CT scan was obtained approximately 13 h after his initial trauma, which showed the earlier left SDH to be significantly decreased in size (Fig. 2).

A new head CT scan performed 2 days later showed complete resolution of the SDH. He progressively became more alert. Approximately 10 days after his admission, a brain MRI study was done which revealed redistribution of the SDH around the left sylvian fissure, the left side of the tentorium and both hemispheral convexities (Fig. 3). The patient

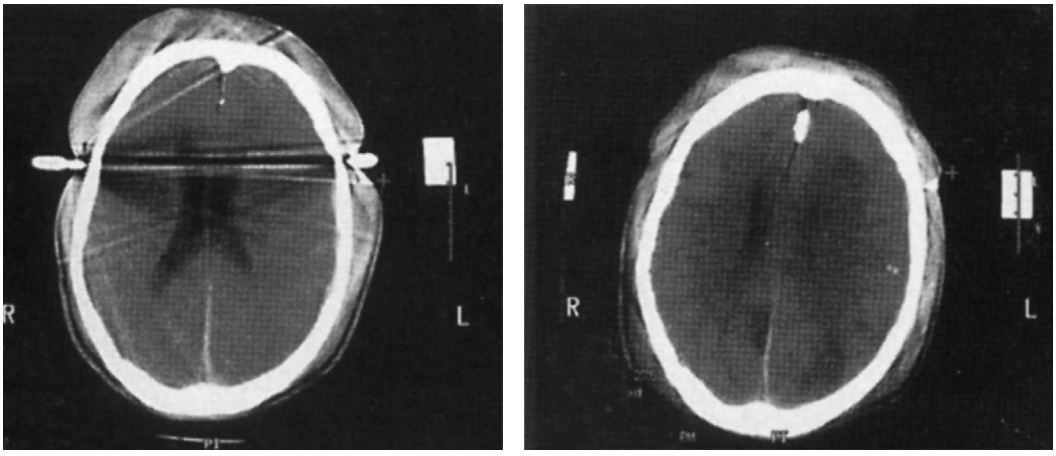

Fig. 2. Head CT scan obtained approximately 6 h later. The previously observed left-sided acute SDH has been significantly decreased in size and the effacement of the ipsilateral ventricle and the midline shift had also been improved.

was finally discharged, 15 days after his admission, without neurological deficits.

1.2. Case 2

Following a major motor vehicle accident, a 24-year-old female was admitted to our emergency room. Her initial GCS score was 7. All her routine blood work were within normal limits. A brain CT scan obtained approximately 1.5 h after her accident showed a rim right-sided, fronto-parietal SDH. Based on the size of the hematoma and the patient's neurological examination conservative management with close follow-up was elected.

The patient was admitted to the neuro-intensive care unit. Four hours after the initial CT scan, another scan was obtained which showed some decrease in the size of the SDH.

Seven days after her admission, a brain MRI study was obtained which showed redistribution of the subdural blood in the right convexity subdural space.

The patient was finally discharged 10 days after her admission with no neurological deficits.

1.3. Case 3

A 29-year-old female was admitted to the emergency room after being involved in a high speed motor vehicle accident. Her GCS score upon admission was 8. Her routine blood work-up were within normal limits. A head CT scan showed a rim right-sided fronto-temporal SDH (9 mm in its largest diameter). The patient was admitted to the neuro-intensive care unit.

Another brain CT scan obtained approximately 6.5 h after her initial scan showed the earlier right-sided SDH to be completely resolved.

The patient progressively became more alert and appropriate and she was finally discharged from the hospital ambulatory and neurologically intact 14 days after her admission.

1.4. Case 4

Following a major motor vehicle accident, a 36-year-old male was admitted to the emergency room. His GCS score was 8. All his routine blood work-up was within normal limits, with the exception of his prothrombin time, which was slightly abnormal. A head CT scan was obtained, which showed a left-sided rim fronto-temporal SDH (approximately 8 mm in its largest diameter).

Based on the size of the hematoma and the patient's neurological examination it was elected for the patient to be conservatively treated. He was admitted to the neuro-intensive care unit. The patient's neurological examination remained unchanged.

Approximately 6.5 h after his trauma, another head CT scan was obtained which showed complete resolution of the left-sided SDH.

The patient progressively became more alert and appropriate. Eight days after his admission, a brain MRI study was obtained, which showed redistribution of the accumulated blood around the sylvian fissure and the tentorium (mainly on the left side) as well as over the convexity of both hemispheres. The patient was finally discharged from the hospital 12 days after his admission.

2. Discussion

Acute SDHs larger than 10 mm in thickness or 5–10 mm thick but associated with deteriorating level of consciousness, pupillary changes, or hemiparesis generally require immediate surgical intervention [1–4,33]. Occasionally, however, spontaneous resolution of acute SDH has been noted within the first 72 post-traumatic hours, obviating the need for operative intervention [7–28]. Numerous cases of spontaneous resolution of SDH occurring between 2 and 72 h after their initial diagnosis have been reported to date in the literature [7–18,21–28].

Various theories have been proposed for explaining the phenomenon of spontaneous resolution of acute cranial SDH. One theory, proposed by Makiyama et al., suggests that the acutely formed SDH is resolved by being forced out of the subdural space via an overlying meningeal tear by the acutely developing brain edema [21]. It has been previously demonstrated that massive brain edema occurs within 20–60 min after a severe head injury [34–36]. The acutely developing brain edema might well be forcing the rapidly accumulated SDH to extradural and extracranial spaces [21]. Niikawa et al. reported one case of acute SDH in which acute brain edema led to spontaneous resolution of the hematoma [25]. In our cases, increased intracranial pressure was documented in one of our patients (Case 1). This finding supports the theory of acutely increased ICP as a driving force responsible for spontaneous resolution of SDH. Occasionally, an associated linear skull fracture can further facilitate the redistribution of the SDH to extracranial spaces. In our series, however, no skull fractures were seen on the obtained CT scans. Lou and Yang reported three cases of acute SDH with rapid spontaneous resolution [37]. Two of their patients also had linear skull fractures and scalp hematomas upon their admission [37]. Interestingly, after the SDH resolution the associated scalp hematomas increased in size, suggesting that part of the SDH moved to the scalp hematomas through a meningeal tear and a skull fracture [37]. Similarly, Kundra and Kundra reported a case in which an acute SDH was spontaneously resolved by moving to extradural spaces through a dural tear and a coronal diastatic fracture, which lead to a corresponding increase in size of the overlying scalp hematoma [19].

Another theory attempting to explain spontaneous resolution of SDH has been proposed by Polman et al. [26]. According to this theory, the accumulated blood clot is diluted and washed out by the circulating cerebro-spinal fluid (CSF) and this liquefied CSF-blood clot mixture is redistributed to

the subdural and subarachnoid spaces [9,16,18,24,26,29]. It has been demonstrated that most cases of acute SDHs are associated with arachnoidal tears permitting the entrance of CSF into the subdural space [29]. The acutely developing post-injury brain edema along with the CSF wash-out effect might well be responsible for the rapid redistribution and resolution of acute SDHs. This explanatory mechanism is further supported by the MRI findings of patients in which spontaneous resolution of an acute SDH occurred [26,29,38]. Polman et al., reported that the obtained brain MRI study demonstrated that the subdural blood had been redistributed over both the cerebral convexities and the tentorium, forming a very thin and sharply demarcated layer [26]. Similarly, Kato et al. reported two cases of spontaneously resolved SDH in which the obtained MRI studies demonstrated redistribution of the hematomas over the cerebral convexity [18]. Likewise, in three of our cases, the obtained MRI studies showed redistribution of the acute SDH along the sylvian fissure, the tentorium and the cerebral convexities. Niikawa et al., in a series of four patients with spontaneous resolution of acute SDH, identified this CSF wash-out mechanism as responsible for hematoma resolution in three of their patients [25]. Further confirmation of this explanatory mechanism is provided by the development of a subacute spinal SDH after the spontaneous resolution of an associated acute cranial SDH [8,11]. Bortolotti et al. and Ahn and Smith have reported two cases in which spinal SDH developed after the rapid spontaneous resolution of cranial SDH [8,11]. Their findings, along with the electron microscopic observation of anatomical continuity of the intracranial and spinal subdural spaces [39,40], further support the theory of redistribution of blood clot-CSF mixture to more dependant areas [8,11].

Accurate prediction of spontaneous resolution of acute cranial SDHs would doubtlessly be of paramount clinical importance. Wu et al., in a previous study, attempted to establish some parameters that could predict spontaneous resolution of acute SDH [29]. They proposed that acute SDHs might spontaneously resolve when their volume is less than 30 mL, they are located in the fronto-temporal or temporo-parietal areas near the sylvian fissure, they demonstrate a characteristic isodense or low dense space between the hematoma and the intracranial wall on CT scan, and they are associated with cerebral tumefaction and/or cerebral contusion and laceration, in neurologically stable patients of young age. It has been suggested that the young brain parenchyma is more elastic and this increased elasticity can facilitate redistribution of the subdural brain clot [11,18,22]. Furthermore, the presence of cortical atrophy is considered a factor that can facilitate spontaneous resolution of acute SDHs [12,13,18,38]. In our series, all the SDHs were smaller than 2 cm in their largest diameters. However, cases of massive SDHs with associated significant midline shift can spontaneously resolve, as has been previously described [13].

Close clinical observation in a neuro-intensive care unit along with ICP and cerebral perfusion pressure (CPP) monitoring (whenever ICP monitoring is indicated according to the Brain Trauma Foundation guidelines [41]) are of paramount importance in these patients that will be conservatively managed. In addition, serial head CT scans are necessary for detecting any changes in the size of the SDH or evolution of any other concomitant brain parenchymal injuries. It is apparent that any deterioration in the clinical neurological examination, abnormalities in the monitored ICP or CPP, or changes in the appearance of the SDH on CT scan represents strong indications for emergent surgical evacuation.

3. Conclusions

The vast majority of acute cranial SDHs larger than 10 mm in thickness require emergent surgical evacuation. However, in rare occasions, redistribution of the SDH in the subdural, subarachnoid or extradural spaces may occur, resulting in spontaneous resolution. Young patients with stable neurological examination who harbor a small volume (<30 mL) SDH located around the sylvian fissure, may be considered as candidates for conservative management. The importance of close clinical follow-up along with ICP and CPP monitoring when this is indicated, and the necessity of obtaining serial head CT scans cannot be overemphasized. Finally, the possibility of the delayed development of spinal acute or subacute SDH in these patients needs to be considered.

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